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The Response of the Dog to Test Doses of Vitamin B_1 Hydrochloride

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Introduction

IN a previous investigation (Worden, Waterhouse & Partington, 1954) dogs were maintained in a metabolism cage on a standard diet and on a known daily water intake and their urinary excretion of vitamin B_1 (aneurine, thiamine) was measured. The volume of urine excreted in each 24-hour period was varied in each of two ways and the quantitative effect on vitamin B_1 excretion and utilisation was measured. The two methods employed were :—

(1) through variation of the standard daily intake of drinking water by giving for the first four to six days a quantity significantly less than the animal would normally consume, followed by six days on the full amount which the animal would consume voluntarily ;

(2) by the use of convection heaters an increase of 30° F. in the environmental temperature was achieved, thereby increasing water loss by evaporation from the animal's body surfaces, with a consequent marked reduction in urine volume.

It was found, as expected, that using method (1) there was a marked decrease in the urinary concentration of vitamin B_1 when the water intake was increased and that as a result the 24-hour output, calculated on the increased volume of urine, remained of the same order. Using method (2) the urine volume decreased at the higher environmental temperature to approximately one-third, but although the vitamin B_1 concentration in this reduced volume was greater, there was a significant drop in the 24-hour urinary excretion of the vitamin.

It was therefore decided to investigate under controlled conditions of dietary intake, water consumption and environmental temperature, the effect of the oral administration to the dogs of vitamin B_1 hydrochloride in a variety of doses. It has already been reported (Worden *et al.*, 1954) that on a similar dietary intake and under similar environmental conditions, the urinary excretion of vitamin B_1 was not proportional to bodyweight. In the present investigation it was decided to employ the same dogs in order to observe any differences that might arise in the effects of comparable dosage with vitamin B_1 on animals of varying breed, sex and bodyweight.

Experimental

The observations were carried out on three dogs (two male; one female) and samples were collected as previously described (Worden, Waterhouse & Partington, 1952, 1954; Worden & Waterhouse, 1955). The identification numbers of the dogs employed in the earlier studies have been retained for convenience.

The various substances were determined in the urine samples by the following methods:—

Vitamin B_1 hydrochloride—the method of Jowett (1940) modified as described in a previous paper (Worden *et al.*, 1952).

Riboflavin—samples containing approximately 15 μg. were acidified with 0.2 ml. hydrochloric acid (36 per cent. w/w HCl) and the mixture extracted with chloroform to remove extraneous soluble material. The aqueous layer was then treated by the procedure described by Kodicek & Wang (1949).

Inorganic phosphate—the method of King (1932).

Urea—a modification of the method of Marshall (1913).

During the experiment each dog received a measured quantity of a diet consisting of pressure-cooked rabbit meat, wholemeal bread and milk. The quantities fed to each dog, together with other individual details, are given in Table I.

Results

Dog No. 1—" Pat "

In the first series in December, 1953, it will be seen that the administration of quantities of 1,000 μg. and 500 μg. of vitamin B_1 hydrochloride was followed in each case by the highest recorded values for daily output of the vitamin in the series. When compared with the day-to-day output, however, the increases were very small and represented a small fraction of the additional vitamin B_1 administered.

In the second series in March, 1954, the administration of 250 μg. was followed by a low 24-hour output (due, apparently, to the small volume of urine voided in this period). The second 24-hour period following administration gave a much larger sample and a consequent increase in vitamin output.

In the third series in May, 1954, the administration of 1,000 μg. produced a significantly higher output in the 24 hours following dosage. The administration of 10,000 μg., an abnormal intake for a healthy animal in positive vitamin B_1 balance, caused a high urinary output in the following 24 hours, and also a higher than normal figure in the next succeeding 24-hour period. After deducting an average amount for the normal daily excretion, however, it willl be seen that less than 1,000 μg. of the 10,000 μg. administered actually appeared in the urine in the four

TABLE I

DETAILS OF INDIVIDUAL DOGS AND THEIR DAILY DIETARY ALLOWANCES

Dog number and name	Breed	Sex	Date of birth	Test number and date	Dietary allowance per 24 hours: Cooked rabbit meat (g.)	Wholemeal bread (g.)	Milk (ml.)	Water (ml.)
1. " Pat "	Mongrel Terrier; smooth-coated	Male	August, 1949	(i) Dec., 1953	227	170	200	500
				(ii) Mar., 1954	227	170	250	750
				(iii) May, 1954	227	170	250	750
4. " Scottie "	Scottish Terrier	Male	June 1950	(i) Nov., 1953	227*	170*	250*	500
				(ii) Feb., 1954	227*	170*	250*	500
				(iii) May, 1954	170	128	188	500
7. " Roxy "	Cocker Spaniel; black and white	Female	April, 1947	(i) Jan., 1954	170	127	188	500
				(ii) Apr., 1954	170	128	188	500
				(iii) July, 1954	170	128	188	850

* A proportion of the daily diet was left uneaten by the dog, and an appropriate allowance has been made for this in the calculations of daily intake.

The vitamin B_1 hydrochloride content of a sample of the homogenised mixed diet was determined each day, by extraction of the vitamin by the method of Kodicek & Wang (1949), followed by application of the method previously described for determination of this vitamin in urine. The riboflavin content of the homogenised diet was determined each day by the method of Kodicek & Wang (1949).

The quantities of vitamin B_1 hydrochloride administered to each animal, in addition to the normal dietary intake during the various series of tests, are shown in Tables II to IV. In each instance the vitamin B_1 was given in the form of an uncoated tablet having a lactose base, and special care was taken to ensure that the dogs did in fact swallow the complete dose.

days following dosage, by which time the 24-hour output had returned to normal for the dietary intake.

Dog No. 4—" Scottie "

In the first series in November, 1953, the administration of 1,000 μg. and 500 μg. of vitamin B_1 hydrochloride caused in each instance a decided increase in vitamin excretion in the 24-hour period following administration. The increase over normal values was in each instance of the order of 150 μg. irrespective of whether 1,000 μg. or 500 μg. had been administered. These increases in output following dosage were in decided contrast to the relatively insignificant increases recorded following a similar experiment (Series 1) with Dog No. 1—" Pat."

In the second series in February, 1954, in the 24 hours following the first dosage with 250 μg. vitamin

TABLE II

THE DIETARY VITAMIN B_1 AND RIBOFLAVIN INTAKE, URINARY VITAMIN B_1 AND RIBOFLAVIN EXCRETION AND OTHER URINARY DATA OF DOGS HOUSED IN METABOLISM CAGES

Dog number and name	Date	Vitamin B_1 intake μg. per 24 hours	Riboflavin intake μg. per 24 hours	Data on urinary samples collected at end of each 24 hours in metabolism cage						
				Volume of sample (ml.)	Specific gravity	Vitamin B_1 μg. per ml.	Vitamin B_1 μg. per 24 hours	Riboflavin μg. per 24 hours	Inorganic phosphate (as P) mg. per 24 hours	Urea g. per 24 hours
1. "Pat"	6.12.53	231	876	—	—	—	—	—	—	—
	7.12.53	207	1,155	363	1·003	0·44	160	248	370	15·1
	8.12.53	159	829	683	1·000	0·25	171	461	468	20·6
	9.12.53	1,215(a)	734	750	1·000	0·32	236	480	586	21·0
	10.12.53	231	931	595	1·000	0·47	277	226	334	14·7
	11.12.53	231	693	703	1·014	0·35	246	309	425	20·0
	12.12.53	223	876	610	1·008	0·26	159	348	298	14·3
	13.12.53	191	1,020	650	1·009	0·32	205	396	516	17·55
	14.12.53	715(b)	1,235	555	1·022	0·32	175	427	662	21·8
	15.12.53	223	995	620	1·014	0·43	264	360	424	18·35
	16.12.53	231	1,075	780	1·018	0·29	222	436	754	20·8
	17.12.53	207	693	690	1·013	0·24	166	372	332	18·4
	18.12.53	—	—	747	1·017	0·25	187	466	614	21·9
	17.3.54	282	1,175	—	—	—	—	—	—	—
	18.3.54	226	976	1,174	1·015	0·21	246	470	684	19·7
	19.3.54	256	985	1,000	1·018	0·21	210	350	460	19·8
	20.3.54	246	1,110	1,060	1·014	0·29	308	752	720	26·9
	21.3.54	237	1,182	870	1·019	0·26	222	740	658	26·6
	22.3.54	462(c)	1,020	800	1·018	0·29	232	607	576	23·6
	23.3.54	244	1,175	580	1·023	0·30	174	852	440	16·2
	24.3.54	246	1,230	1,310	1·023	0·28	367	1,205	960	38·9
	25.3.54	2,232(d)	1,060	920	1·017	0·21	194	764	450	23·2
	26.3.54	256	1,090	970	1·019	0·62	602	689	662	32·8
	27.3.54	228	1,060	737	1·008	0·30	221	612	346	22·5
	28.3.54	237	1,138	930	1·021	0·24	224	642	672	25·2
	29.3.54	—	—	666	1·020	0·30	200	586	496	22·0
	24.5.54	395	1,862	—	—	—	—	—	—	—
	25.5.54	259	1,528	620	1·010	0·39	242	310	218	11·35
	26.5.54	241	1,555	1,015	1·017	0·29	289	446	792	25·6
	27.5.54	248	1,830	710	1·013	0·31	220	340	322	17·25
	28.5.54	1,285(e)	1,700	1,105	1·018	0·26	282	720	773·5	29·6
	29.5.54	274	1,700	1,010	1·015	0·41	409	525	611	29·0
	30.5.54	274	1,425	698	1·002	0·20	140	238	163	10·5
	31.5.54	274	1,260	808	1·006	0·25	202	580	505	20·8
	1.6.54	10,284(f)	1,630	870	1·016	0·26	226	652	612	15·7
	2.6.54	241	2,194	1,020	1·009	0·82	836	560	520	26·3
	3.6.54	230	1,335	920	1·006	0·44	405	524	418	22·8
	4.6.54	219	1,250	890	1·014	0·29	258	454	487	20·2
	5.6.54	—	—	890	1·013	0·29	258	410	486	23·5

(a) 1,000 μg. vitamin B_1 hydrochloride administered 10 a.m. 9.12.53, in addition to vitamin B_1 hydrochloride in diet.
(b) 500 μg. " " " 14.12.53, " " " " "
(c) 250 μg. " " " 22.3.54, " " " " "
(d) 2,000 μg. " " " 25.3.54, " " " " "
(e) 1,000 μg. " " " 28.5.54, " " " " "
(f) 10,000 μg. " " " 1.6.54, " " " " "

B_1 hydrochloride the dog, for some inexplicable reason, voided only 190 ml. urine, and the 24-hour vitamin B_1 hydrochloride output was 76 μg., a result less than on the day before dosing. In the next 24-hour period, however, the dog voided 530 ml. urine and the total vitamin B_1 hydrochloride output was 228 μg. Following the 250 μg. dose, a dose of 2,000 μg. was administered four days later and the 24-hour output of vitamin B_1 hydrochloride for both the subsequent two days was considerably increased.

In the third series in May, 1954, the administration of 1,000 μg. produced no really significant increase, either in 24-hour vitamin output, or in concentration of vitamin B_1 hydrochloride per ml. of urine. The administration of 10,000 μg. of vitamin B_1 hydrochloride produced, as expected, a high output of vitamin in the following 24-hour period. The amount of vitamin voided in the urine, over and above that normally excreted from the diet was, however, only some 400 to 500 μg. of the 10,000 μg. actually administered, an even lower proportion than in the case of Dog No. 1—"Pat."

Dog No. 7—"Roxy"

In the first series in January, 1954, the administration of 1,000 μg. of vitamin B_1 hydrochloride was followed by an increase of approximately 100 μg. in the excretion of vitamin during the succeeding 24-hour period, and the administration of 500 μg. was followed by an increase of approximately 50 μg. Thus Dog No. 7 was the only animal of those used which gave a detectable and quantitative response to these relatively lower dosages employed in the first series for each dog.

TABLE III

THE DIETARY VITAMIN B_1 AND RIBOFLAVIN INTAKE, URINARY VITAMIN B_1 AND RIBOFLAVIN EXCRETION AND OTHER URINARY DATA OF DOGS HOUSED IN METABOLISM CAGES

Dog number and name	Date	Vitamin B_1 intake μg. per 24 hours	Riboflavin intake μg. per 24 hours	Data on urinary samples collected at end of each 24 hours in metabolism cage						
				Volume of sample (ml.)	Specific gravity	Vitamin B_1		Riboflavin	Inorganic phosphate (as P)	Urea
						μg. per ml.	μg. per 24 hours	μg. per 24 hours	mg. per 24 hours	g. per 24 hours
4. "Scottie"	22.11.53	214	885	—	—	—	—	—	—	—
	23.11.53	191	856	492	1·012	0·29	140	206	397	12·3
	24.11.53	237	1,132	520	1·005	0·26	135	213	388	15·1
	25.11.53	1,254(a)	1,060	700	1·009	0·28	196	217	465	17·8
	26.11.53	246	880	560	1·004	0·56	314	202	384	16·9
	27.11.53	196	812	580	1·000	0·37	212	203	339	15·3
	28.11.53	214	878	475	1·004	0·29	138	190	378	15·7
	29.11.53	230	790	540	1·009	0·32	170	227	400	19·1
	30.11.53	745(b)	1,100	610	1·007	0·26	156	292	480	20·2
	1.12.53	197	760	666	1·007	0·52	346	326	562	19·25
	2.12.53	208	1,000	534	1·002	0·28	147	288	313	18·7
	3.12.53	271	950	600	1·018	0·21	126	330	430	17·8
	4.12.53	—	—	570	1·005	0·21	120	445	368	18·1
	15.2.54	179	960	—	—	—	—	—	—	—
	16.2.54	146·5	822	416	1·013	0·24	100	229	325	3·26
	17.2.54	160	573	488	1·014	0·23	110	293	388	6·14
	18.2.54	394(c)	816	412	1·019	0·25	101	248	321	8·45
	19.2.54	172	695	190	1·021	0·50	76	159·5	179	4·35
	20.2.54	141	700	530	1·028	0·43	228	504	424	5·91
	21.2.54	130	535	400	1·023	0·23	92	244	254	7·25
	22.2.54	110	556	330	1·014	0·29	96	218	284	7·05
	23.2.54	2,252(d)	972	324	1·015	0·26	84	226	285	7·38
	24.2.54	167·5	815	350	1·022	0·83	290	382	403	9·90
	25.2.54	140	630	556	1·034	0·46	256	348	480	12·90
	26.2.54	173·5	695	440	1·019	0·30	132	189	308	8·58
	27.2.54	—	—	440	1·015	0·27	119	207	373	11·00
	10.5.54	199	890	—	—	—	—	—	—	—
	11.5.54	159	800	280	1·021	0·41	115	145·5	198	8·0
	12.5.54	165	824	405	1·018	0·43	174	178	218	10·3
	13.5.54	192	1,165	515	1·029	0·46	237	284	403	18·7
	14.5.54	1,168(e)	746	670	1·013	0·41	275	275	438	22·4
	15.5.54	206	768	455	1·012	0·43	196	236	250	12·75
	16.5.54	171·5	631	580	1·013	0·34	197	266	352	16·3
	17.5.54	178·5	890	540	1·019	0·39	210	351	486	18·25
	18.5.54	10,212(f)	890	320	1·016	0·42	131	304	142	10·5
	19.5.54	220	775	590	1·015	0·98	578	395	381	15·7
	20.5.54	178	856	536	1·020	0·45	241	262	375	17·0
	21.5.54	247	974	565	1·020	0·45	254	288	464	19·8
	22.5.54	—	—	440	1·013	0·44	194	154	283	12·1

(a) 1,000 μg. vitamin B_1 hydrochloride administered 10 a.m., 25.11.53, in addition to vitamin B_1 hydrochloride in diet.
(b) 500 μg. " " " 30.11.53 " " " "
(c) 250 μg. " " " 18.2.54, " " " "
(d) 2,000 μg. " " " 23.2.54, " " " "
(e) 1,000 μg. " " " 14.5.54, " " " "
(f) 10,000 μg. " " " 18.5.54, " " " "

In the second series in April, 1954, this dog continued to show a greater response to these dose levels of vitamin B_1 hydrochloride than did either of the other animals. Administration of 250 μg. caused a vitamin excretion in excess of the normal level on the standard diet of approximately 220 μg. during the following 24-hour period. Thus in this instance alone, among all those studied, was an amount of vitamin equivalent to almost the whole of that administered, excreted in the urine within 24 hours. The administration of 2,000 μg. of vitamin B_1 hydrochloride resulted in a high rate of excretion for no less than three succeeding 24-hour periods. The figures for vitamin excretion, after deducting the normal output on the standard diet, were approximately 620 μg., 200 μg. and 120 μg. in the succeeding three 24-hour periods. Here again, virtually half the vitamin administered was subsequently voided in the urine—a proportion much greater than was obtained with either of the other dogs at this dose level.

In the third series in July, 1954, the administration of 1,000 μg. of vitamin B_1 hydrochloride caused an increase in 24-hour output of only approximately 35 μg. This was the only instance when Dog No. 7 gave so low a response to dosage. Following the administration of 10,000 μg. of vitamin B_1 hydro-

TABLE IV

THE DIETARY VITAMIN B_1 AND RIBOFLAVIN INTAKE, URINARY VITAMIN B_1 AND RIBOFLAVIN EXCRETION AND OTHER URINARY DATA OF DOGS HOUSED IN METABOLISM CAGES

Dog number and name	Date	Vitamin B_1 intake μg. per 24 hours	Riboflavin intake μg. per 24 hours	Data on urinary samples collected at end of each 24 hours in metabolism cage						
				Volume of sample (ml.)	Specific gravity	Vitamin B_1		Riboflavin μg. per 24 hours	Inorganic phosphate (as P) mg. per 24 hours	Urea g. per 24 hours
						μg. per ml.	μg. per 24 hours			
7. "Roxy"	18.1.54	189·5	846	—	—	—	—	—	—	—
	19.1.54	174·5	1 048	560	1·009	0·17	95	336	191	9·4
	20.1.54	183·5	1 040	422	1·029	0·26	110	264	455	11·3
	21.1.54	1 167(a)	867	666	1·013	0·22	146·5	645	706	15·3
	22.1.54	173·5	735	664	1·012	0·40	265·5	584	527	16·6
	23.1.54	42·5	174	265	1·015	0·31	82	304	210·5	7·9
	24.1.54	169·5	995	590	1·021	0·18	106	614	491	16·7
	25.1.54	224	1 130	540	1·006	0·17	89	890	348	10·4
	26.1.54	715·5(b)	975	614	1·018	0·21	126	485	441	14·2
	27.1.54	203·5	624	355	1·015	0·48	172	262	287	10·4
	29.1.54	173·5	807·5	350	1·031	0·39	137	396	378	11·2
	29.1.54	187	614	835	1·014	0·18	150	501	433	21·4
	30.1.54	—	—	474	1·017	0·24	114	326	368	15·2
	21.4.54	193	692	—	—	—	—	—	—	—
	22.4.54	192	617	600	1·009	0·28	168	282	339	11·9
	23.4.54	199	857	510	1·003	0·27	135	204	216	9·95
	24.4.54	456(c)	714	646	1·007	0·28	178	300	404	15·5
	25.4.54	199	940	805	1·010	0·49	394	466	916	26·8
	26.4.54	165	918	336	1·012	0·30	101	351	210	10·09
	27.4.54	178	768	650	1·016	0·31	202	650	611	21·4
	28.4.54	186	1 000	500	1·014	0·32	157·5	535	450	16·85
	29.4.54	2 182·5(d)	1,048	650	1·016	0·29	188·5	650	558	22·0
	30.4.54	160	831	700	1·012	1·17	820	624	546	21·85
	1.5.54	192	1,062	584	1·013	0·69	404	520	412	17·4
	2.5.54	199	822	720	1·013	0·45	324	612	648	21·6
	3.5.54	—	—	300	1·010	0·49	147	474	217·5	9·9
	29.6.54	173	636	—	—	—	—	—	—	—
	30.6.54	118	1,020	468	1·009	0·15	70	150	334	9·3
	1.7.54	148	590	416	1·009	0·12	47·8	166	294	9·9
	2.7.54	165	724	740	1·017	0·11	81·4	288	510	19·8
	3.7.54	1,165(e)	660	670	1·011	0·13	87	268	486	19·4
	4.7.54	130	625	574	1·014	0·21	120	230	348	14·1
	5.7.54	140	703	696	1·011	0·12	83·5	320	403	14·8
	6.7.54	157	825	352	1·019	0·13	46·8	272	545	12·1
	7.7.54	10,126(f)	700	546	1·014	0·13	71	311	356	16·3
	8.7.54	133	580	466	1·019	0·98	456	261	372	13·9
	9.7.54	157	754	572	1·018	0·49	280	320	406	15·8
	10.7.54	173	660	672	1·017	0·35	235	336	420	16·2
	11.7.54	—	—	536	1·019	1·83	980	289	366	14·1

(a) 1,000 μg. vitamin B_1 hydrochloride administered 10 a.m., 21.1.54, in addition to vitamin B_1 hydrochloride in diet.
(b) 500 μg. " " " 26.1.54 " " " "
(c) 250 μg. " " " 24.4.54 " " " "
(d) 2,000 μg. " " " 29.4.54 " " " "
(e) 1,000 μg. " " " 3.7.54 " " " "
(f) 10,000 μg. " " " 7.7.54 " " " "

chloride this dog gave a most unusual response. After deducting the normal vitamin excretion when on standard diet, the excretion in the four 24-hour periods following dosage was approximately as follows: 380 μg., 200 μg., 160 μg., 900 μg. The high rate of excretion on the fourth day was very unusual, but unfortunately the animal had, in accordance with normal routine, been removed from the cage before the analysis could be completed and therefore no further values were available for subsequent days.

Discussion

The urinary excretion of vitamin B_1 by human beings has been studied in considerable detail and a method of assessing nutritional status, known as the Test-Dose Method, was introduced by Harris & Leong (1936). The concentration of vitamin B_1 in samples of urine both before and after a test dose was estimated by the thiochrome method. In cases of B_1 deficiency only a small proportion of the dose was excreted, compared with a relatively large proportion in cases known to be in positive vitamin B_1 balance.

Wang & Harris (1939) obtained an average value of 65 μg. for the daily urinary output by human beings, and this rose to 105 μg. following administration of a test dose of 1 mg. Average daily values of below 30 μg. were considered to indicate subnormal levels of intake and below 15 μg. were considered as indicating severe B_1 deficiency.

The test-dose method has been extensively used for a variety of doses under a variety of experimental conditions and the urinary output has been determined within 24 hours or within a period as little as three or four hours of the test dose.

Melnick, Field & Robinson (1939) found an average value for daily excretion to be 198 μg. for men and 93 μg. for women. After administering 5 mg. of vitamin B_1 daily, he found an excretion of 14 per cent. of test dose for men and 12 per cent. for women within 24 hours of dosing. Numerous other values have been quoted for the daily excretion of vitamin B_1 in the urine by human subjects under a variety of conditions and their responses to various levels of test doses measured.

Sure & Ford (1943) found that in rats doses of 5 to 50 μg. of vitamin B_1 per day were the levels best utilised. They found the efficiency, based on vitamin B_1 excreted in the urine, for doses of 50, 100 and 1,000 μg. to be 92, 83 and 52 per cent. respectively.

More recently, however, some doubt has arisen as to the reliability of the test-dose method. Allbone & Finch (1945) found that 10 to 14 per cent. of their intake was excreted in the urine by children on a normal diet. Following an oral dose of 0.5 to 1.0 mg., the output varied by 0 to 41 per cent. of the intake, but the average was still 14 per cent.

Mickelson, Caster & Keys (1946, 1947) made a statistical study of the values for vitamin B_1 excretion by groups of young men using the test-dose method. With daily dietary intakes of 600 μg., 1,000 μg., 1,800 μg. and 2,000 μg. the amount of vitamin B_1 excreted in the urine—determined by the thiochrome method—was linearly related to the intake. Individual excretions on similar diets, however, varied considerably, and both individual and group variations increased as the amounts excreted increased. These authors also investigated the excretion of the pyrimidine portion of the molecule and found that it bore an exponential relationship to the vitamin B_1 intake. They considered that at vitamin B_1 intakes from 700 μg. to 1,000 μg. per day, the vitamin B_1 excretion is the better indication of nutritional status, but that outside these limits, the pyrimidine excretion is the better indication.

In studying response to test doses of vitamin B_1 in any animal species, consideration must be given to a possible failure to absorb the whole of the test dose. Unfortunately a determination of the vitamin B_1 content of the faeces does not give reliable information on this point. Emerson & Obermeyer (1945) found that, using rats, the amount of vitamin B_1 (present largely as cocarboxylase) in the faeces was constant whether the rats received 0, 5 or 50 μg. daily, although in deficient animals the volume of faeces was much reduced. It has been shown by a number of workers that the faecal excretion of vitamin B_1 is relatively constant and is independent of dietary intake, and that most of the vitamin B_1 in the faeces is produced by bacterial synthesis (Wildermann, 1941; Alexander, Landwehr & Mitchell, 1946). Kirk & Chieffi (1951), however, stated that from 50 to 75 per cent. of vitamin B_1 administered in tablet form was excreted in the faeces.

In experiments designed to test the response of the dog to administration of test doses of vitamin B_1 there are, of course, certain factors which do not arise in experiments with human patients. This particularly concerns the collection of samples, for which purpose it is essential to use a suitable apparatus, such as a metabolism cage. It is also not possible to collect a complete specimen of urine at the end of a predetermined number of hours without employing catheterisation, and this is considered by many workers to be undesirable as interfering with normal physiological processes (Worden, 1939). In employing a method involving the examination of samples representing 24-hour periods both before and after dosing, great care must be exercised in interpreting the results obtained. It has already been emphasised (Worden *et al.*, 1952, 1954, 1955) that, in this kind of work, results quoted for vitamin excretion by the dog in 24-hour periods must be related to the values obtained over several such consecutive periods. This is difficult, however, in work involving administration of test doses of vitamin B_1 where, in most cases, the whole of the resulting urinary excretion occurs in the two following 24-hour periods.

The results given above for Dog No. 4—"Scottie"—in the second series of tests in February, 1954, illustrate this point. On the day following administration of 250 μg., the 24-hour urine excretion actually fell to 76 μg., whereas prior to dosing it has been 100, 110 and 101 μg. on three consecutive days. This was due solely to the fact that the volume of urine voided by the dog on that day fell to 190 ml. from a previous average of about 400 ml. per day. On the following day, the second 24-hour period after dosing, the dog voided 530 ml. of urine and the excretion of vitamin B_1 hydrochloride rose to 228 μg. This illustrates the difficulty in assessing the results in this type of test, and shows the errors which may result in taking results over a fixed period. A retention of urine by the animal, or an irregular voiding of the urine, may greatly influence any figures for 24-hour output. In the particular instance quoted, in fact, the effect of dosing would seem to be best illustrated by reference to the increase in concentration of vitamin B_1 hydrochloride per ml. of urine throughout the 48 hours following dosing.

To complicate the position further, there is the behaviour of Dog No. 7—"Roxy"—in the third series of tests carried out in July, 1954. In this instance, following a dose of 10,000 μg., the dog voided substantially increased amounts of vitamin B_1 in the 24-hour urine samples for as long as four days, there being a dramatic further rise on the fourth day. We are not aware of any published work in which a similar finding has been recorded.

Our observations on only a very few animals indicate that there are marked individual variations in response to test doses of vitamin B_1, and that it is extremely unlikely that any simple procedure based on this principle would yield significant results in clinical practice. They are in keeping with our earlier findings that the utilisation and excretion of this vitamin are not necessarily proportional to bodyweight but are influenced by temperament and activity. Early studies by Himwich, Goldfarb & Cowgill

(1932) showed that increased amounts of vitamin B_1 were required by dogs to which thyroid gland was fed, and later work on other species has confirmed that the vitamin is utilised more rapidly when the metabolic rate is raised (see Rosenberg, 1942, pp. 144-145). Modern concepts of the metabolism of vitamin B_1 suggest that it is rapidly utilised following absorption, and that during growth, at least, it needs to be provided at all times in order to facilitate optimal utilisation of the total diet. Thus, in their studies on young chicks, Waibel, Cravens, Bird & Baumann (1954) have shown that the more frequently vitamin B_1 is injected the more efficiently is it utilised, administration twice daily being superior to once daily or to less frequent administration.

The effect of administering vitamin B_1 does not appear to have exerted any consistent effect upon the urinary excretion of riboflavin. The urinary excretion of inorganic phosphate was likewise not affected by the level of vitamin B_1 administered or excreted, but it again bore the remarkable parallel to urinary riboflavin output noted in earlier studies (Worden & Waterhouse, 1955).

Summary

1. Three dogs of varying breed, sex and bodyweight have been maintained in a metabolism cage and provided with standard diets and a standard water intake, and in these circumstances the response to test doses of vitamin B_1 hydrochloride, varying from 250 to 10,000 μg., has been studied.

2. A marked individual variation in the response to these test doses has been observed, and in one instance only out of 18 has an amount of vitamin B_1 equivalent to virtually the whole of the test dose been excreted in the urine over the following 24 hours.

3. At higher test dosage levels, the urinary level of vitamin B_1 has been increased over 48 hours and in one instance over 96 hours.

4. Variations in urinary volume exert a marked effect upon the amount of vitamin B_1 excreted per 24 hours, there being considerably less fluctuation in the urinary concentration than in the total amount excreted over a 24-hour period.

5. There is little indication from these studies that any simple form of test dosage would be of value in veterinary clinical practice in establishing the vitamin B_1 status of dogs. Observations over more than one day are necessary in order to establish the effect of a single test dose.

6. The administration of doses of up to 10,000 μg. of vitamin B_1 hydrochloride have not been found to exert any significant effect upon the urinary excretion of either riboflavin or inorganic phosphate. The urinary excretions of phosphorus and riboflavin again showed the remarkable parallel which has been previously noted.

Acknowledgments.—We are again grateful to Dr. E. Kodicek, of the Dunn Nutritional Laboratory, Cambridge, for his interest and advice, and to our colleague, Mr. J. Bunyan, for assistance with certain aspects. We are indebted also to Miss M. Hardaker and Mr. M. J. Rix for some of the analyses and for care of the experimental animals.

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CCTA/WHO TRAINING COURSE ON RABIES

After several years of negotiation by IBED (Interafrican Bureau of Epizootic Diseases) arrangements have been completed between CCTA representing the Governments of U.K., France, Belgium, Portugal, Union of South Africa, and Federation of Rhodesia and Nyasaland on the one side and WHO on the other, for the holding of three weeks laboratory technique training course in rabies at EAVRO laboratories Muguga, Kenya, from July 11th to 28th, 1955.

Thirty-five attendees have been invited from all over Africa and will be instructed by such well-known personalities in the rabies field as Kaplow, Koprowski, Komarov, Lepine and Habel. Further details can be obtained from Mr. W. G. Beaton, Interafrican Bureau of Epizootic Diseases, Muguga, P.O. Kikuyu, Kenya, who is the organising secretary of the Course.

INTERAFRICAN ADVISORY COMMITTEE ON EPIZOOTIC DISEASES OF AFRICA (IACED)

This Committee will meet at Muguga from July 5th to 8th, 1955. Special consideration is being given to the subjects of Vaccination in Foot-and-Mouth Disease in Africa, Complement Fixation Test in Diagnosis of Contagious Bovine Pleuro-pneumonia and Bovine Cutaneous Streptothricosis. Mr. W. G. Beaton is secretary to the Committee.

Curled Tongue in Turkey Poults

BY
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Introduction

A DEFORMITY of the tongue in young turkey poults characterised by a curling or folding back of the distal end of the tongue on to the dorsal surface was first recorded by Hudson (1939) in the U.S.A. Field enquiries indicated that the condition, termed "Curled Tongue," was fairly widely distributed throughout the main turkey-producing areas in the United States. Relatively few affected birds died and economic loss was sustained mainly through retarded growth rate due to the inability of the birds to feed properly.

Sporadic cases of curled tongue in poults have been noted in recent years in Britain both on farms and in material submitted to veterinary laboratories for *post-mortem* examination. The evidence in such cases indicates that relatively few birds in a flock are affected.

The aetiology of the condition remains somewhat obscure. Grau & Almquist (1945) describe a similar condition encountered in chickens fed simplified diets and caused by a dietary deficiency of the essential amino acids, isoleucine, leucine and phenylalanine. The varied protein contributions of the ingredients of practical chick and turkey starter diets are unlikely to result in quantitative inadequacy of these particular amino acids. This is supported by the work of Scott (1951) and Bragg (1953) who concluded that under normal growing conditions, dietary protein is not a contributory factor in the cause of curled tongue in turkey poults.

Bragg (loc. cit.) suggests a genetic basis of the condition and postulates that "this may be explained by a fairly rare recessive genetic factor or a genetic difference in the nutritional requirements of the breeder hens in the flock supplying hatching eggs." This conjecture is supported, however, on the meagre evidence of five out of 2,442 poults examined at hatching time which showed deformed tongues. Further, it was not established by histological examination whether the tongue condition noted in the newly hatched poults was identical to that which developed in normal poults during the subsequent brooding period.

In the same series of experiments, the addition of the antibiotics bacitracin or terramycin was ineffective in preventing curled tongue. All diets were of dry mash fed *ad libitum* and a greater incidence of the trouble was experienced among poults fed mash of fine texture than among those fed a coarser type diet of similar composition. Bragg cites personal communications arising from field observations which lend support to the view that fineness of division of the feed is a critical factor in the prevention of curled tongue.

In the experiments carried out by Scott (loc. cit.) high energy diets were fed to the poults during the early weeks of life. All mash diets rich in wheat flour pasted up in the beaks of the poults and caused the tongue to curl back mechanically. When the level of flour was reduced or replaced by maize meal, ground oats or wheat middlings or a combination of these ingredients, the texture of the complete mash was much coarser and no incidence of curled tongue occurred.

Experimental

About 1,000 turkeys are reared each year at the National Institute of Poultry Husbandry. These comprise various breeds and crosses produced from adult stock maintained on the plant. Curled tongue had not been observed at the Institute prior to 1954 but in June of that year typical symptoms of the deformity were reported in three poults submitted for *post-mortem* examination.

At that time 450 live poults varying in age from four to 39 days were available for individual examination and of these, 79 birds (17·6 per cent.) were found to be affected. The extent of tongue curling and the point at which it occurred varied somewhat, but in most cases the retraction was immediately anterior to the arrow-shaped head of the entoglossal bone. A typical case of curled tongue is shown in Plate 1. The mucous membrane of the mouth appeared to be very dry and particles of feed had accumulated at either side and beneath the tongue of some of the affected birds. The only external evidence of any abnormality was a slight swelling below the lower mandible but this was not a consistent finding. Curled tongue mainly appeared in poults during the first 14 days of life.

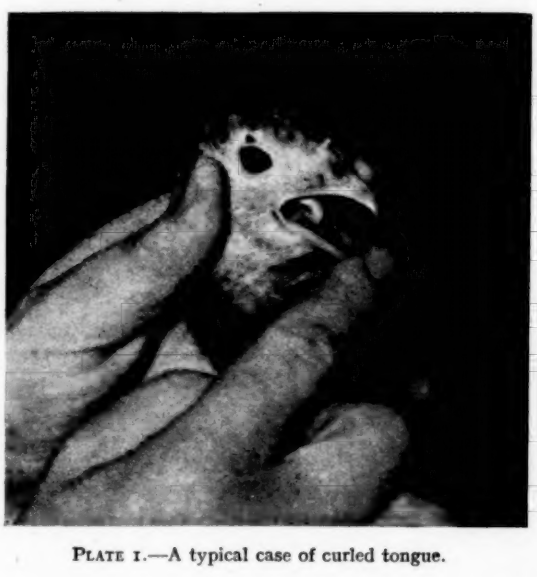

PLATE 1.—A typical case of curled tongue.

Through the co-operation of Mr. H. E. Roberts, Assistant V.I.O. for the West Midland Province, a limited number of sections were prepared of typical curled tongues. These did not reveal any gross histopathological changes when compared with sections of tongues taken from poults which had died from other conditions. The nature of the deformity remains obscure but the evidence of the experiments which follow indicate a purely mechanical origin.

For the first two weeks after hatching the poults were accommodated in tier brooders fitted with contact heater pads and thereafter in compartments of a solid floor house where they were brooded under electrically heated hovers and had access to sun parlours. At six weeks of age the poults were transferred to unheated solid floor compartments in an intensive rearing house where they remained until 12 weeks of age.

From day old the poults were allowed free access to a home-mixed dry mash and to a supply of insoluble grit. The composition of the mash was as follows :—

Parts by Weight	
21·75	Yellow maize meal
25·00	Ground wheat
5·00	Wheat bran
10·00	Wheat middlings
5·00	Grass meal
8·00	Decorticated groundnut meal
10·00	White fish meal
8·00	Meat and bone meal
3·00	Dried brewer's yeast
1·50	Steamed bone flour
2·00	Ground limestone
0·50	Common salt
0·25	Synthetic vitamin D_3 supplement
100·00	

The system of brooding and rearing was similar to that employed in 1953 and the composition of the diet was identical save that a source of synthetic vitamin D_3 was used in place of cod-liver oil. The latter had been included at a 2 per cent. level and although the texture and appearance of the mixed mash seemed satisfactory, the omission of the oil may have resulted in a slightly drier consistency.

Immediately the extent of curled tongue had been determined in the poults comprising the first six hatches, the system of feeding was changed to wet mash, but no alteration was made in the composition of the diet. Regular examinations were made of the progress of these groups and, irrespective of the age of the birds at the time of changing to wet mash feeding, after a lapse of seven days, no new cases of curled tongue appeared. A total of 86 poults, representing 16·4 per cent. of the original number of birds in the first six hatches developed curled tongue. This is possibly an underestimate of the incidence for it excludes turkey chicks which died before the condition was recognised and which may have had deformed tongues.

In the earlier hatched groups the rate of growth had undoubtedly been checked, feathering was unkempt and the birds looked generally unthrifty. Their debility affected the interpretation of the subsequent progress of these groups for the poults suffered heavy mortality through coccidiosis. On *post-mortem* examination, however, apart from a few cases of asphyxiation there was no evidence to suggest that the curled tongue condition *per se* had been responsible for death. On the contrary, the majority of birds which at some earlier age had shown curled tongue, subsequently recovered and by 12 weeks of age only six poults were still affected.

The beneficial effects resulting merely from a change in the physical form in which the food was offered did not suggest that dietary deficiencies were responsible for the curled tongue condition. A review of the available literature does not provide any evidence of the normal requirements of turkey poults for phenylalanine, leucine or isoleucine, but the National Agricultural Research Council of America (1950) place the minimum requirements of young chickens for these amino acids at 0·7, 1·4 and 0·5 per cent. respectively of the total diet.

Based on tables of average composition, the home-mixed turkey starter diet used in the present instance contained 1·4, 1·2 and 2·0 times the normal baby chick requirements for phenylalanine, leucine and isoleucine respectively.

As a further check on the nutritive adequacy of the diet, a subsequent hatch of poults (7), was subdivided at hatching, one lot being started in tier brooders for two weeks and then moved to solid floor conditions, and the other brooded throughout in pens with solid floors. A purchased turkey starter mash was fed dry and *ad lib.* to both groups. Curled tongue appeared in 19 per cent. of the poults which were tier brooded but no cases were observed in the floor-brooded group.

The sudden incidence of curled tongue in three separate breeds and in one cross following the complete absence of any deformity of the tongue in the poults of preceding generations pointed to other than a genetical origin. Further, all poults produced after the discovery of the trouble were examined at hatching and in no case was any abnormality of the tongue present. The housing of the breeding stock was identical with that in the preceding year. With the exception of the Broad-breasted Bronze, which had a grass run, the birds were housed in straw yards.

During 1953 and preceding years the breeding stock were fed purchased diets of undisclosed composition. In 1954 a diet of grain and grain balancer breeder's mash was fed in approximately equal proportions by weight. The composition was as follows :—

Parts by Weight		
	Mash	*Grain*
10	Maize meal	Equal parts wheat
10	Bran	Barley
30	Middlings	
30	Ground oats	
10	Fish meal	
7	Dried grass meal	
3	Dried yeast	
100		

The mash was supplemented with 0·5 per cent. of a synthetic source of vitamin D_3 with a potency of 500 B.S.I. units of vitamin D_3 per gramme.

A group (8) of day-old Broad-breasted Bronze and Broad-breasted White poults was purchased from a local breeder to test the adequacy of the maternal diet and to explore further the possible influence of inherent factors. These were produced from stock housed in straw yards and fed an entirely different diet to that used for our own breeding birds. Further, no cases of curled tongue had appeared in the growing stage in the progeny hatched earlier in the season from the same breeding stock. On arrival the poults were subdivided according to breed, one group being started in tier brooders for the first 14 days and the other group floor brooded from day old. Both groups had free access to the home-mixed starter diet fed dry and to a supply of insoluble grit. No deformity of the tongue was apparent when the poults were received and none of the Broad-breasted White poults subsequently showed any abnormality. Of the Broad-breasted Bronze poults started in tier brooders and on solid floors, 18·5 and 14·3 per cent. respectively developed typical curled tongues.

The individual ingredients of the home-mixed starter mash were purchased at intervals of approximately one month and considerable variation could have occurred between consignments in the fineness of grinding, particularly of the cereal components. To examine this point a further hatch (9) of home-bred poults was subdivided at day old for brooding in tiers or on solid floors. The two groups were allowed free access to a dry mash diet compounded to the same formula as that used for the earlier hatches but mixed from a new delivery of individual components. Curled tongue occurred in the first few weeks of life among poults in both groups.

The only modification in the brooding and feeding arrangements for a hatch of poults seven days later (10), was the provision of dry mash for those accommodated in the tier brooders and of wet mash for the group which was floor brooded. Cases of curled tongue developed only in the group fed dry mash.

Two subsequent hatches were again subdivided and brooded either in tiers or on solid floors. All four groups were fed wet mash but the home-compounded mixture was used for one hatch (11) and a purchased turkey starter mash for the other (12). Only one poult developed curled tongue and this occurred in the group fed the purchased mash and floor brooded.

A representative sample from a batch of home-compounded starter mash was submitted for physical examination. The consignment from which it was drawn had given rise to pronounced curled tongue when fed in a dry state. The results of the examination shown in Table I are compared with those relating to four other turkey starter mashes which had been used in Scotland. A fairly high incidence of curled tongue had been observed with three of these mashes (A, B and C) when fed dry, whereas very few poults reared on Mash D were affected.

TABLE I

PHYSICAL COMPOSITION OF TURKEY STARTER MASHES

	Percentage of Weight as Particles	
	Above 1 m.m.	Below 1 m.m.
N.I.P.H. Experimental Mash	10·5	89·5
Mash A	26·6	73·4
Mash B	13·2	86·8
Mash C	26·1	73·9
Mash D	42·0	58·0

At least 70 per cent. of the weight of the mashes which gave rise to curled tongue comprised particles below 1 mm. in diameter, and in the case of the N.I.P.H. experimental mash this value was nearly 90 per cent. Mash D, in contrast, had a much higher proportion of coarse particles.

Finally, two further hatches of poults (13 and 14) separated by an interval of seven days, were accommodated in tier brooders and on solid floors respectively. Each hatch was subdivided according to breed, one group being fed the home-mixed starter diet dry and the other group as moist mash. Curled tongue occurred only in poults of the first hatch receiving dry mash.

The results of all the tests are summarised in Table II.

TABLE II

PERCENTAGE OF INITIAL POULTS WHICH DEVELOPED CURLED TONGUE (All breeds)

Hatch No.	Treatment	Tier brooded to 14 days Floor brooded thereafter		Floor brooded throughout	
		Dry Mash fed	Wet Mash fed	Dry Mash fed	Wet Mash fed
1–6	Home compounded diet	16·4			
7	Purchased turkey mash	19·0		Nil	
8	Home compounded diet Purchased poults	9·8		8·3	
9	Home compounded diet	7·5		8·2	
10	Home compounded diet	26·3			Nil
11	Home compounded diet		Nil		Nil
12	Purchased turkey mash		Nil		2·9
13	Home compounded diet	10·7	Nil		
14	Home compounded diet			Nil	Nil
7–14		13·9	Nil	4·9	0·7

Throughout the whole series of experiments the incidence of curled tongue tended to be higher in poults reared for the first two weeks of life in tier brooders. The water supply in the particular model used is some distance from the heated brooder compartment and during the first few days of life the intake of water by the poults may have been limited. Further, a consistently higher room temperature is maintained with wire-floored tier brooders compared with a solid-floor brooder house employing electrically-heated hovers. A high ambient temperature combined with a sub-optimal intake of water is likely to aggravate the effects of dry mash feeding in promoting the curled tongue condition. That the system of brooding could only have exerted a secondary effect is evident from the fact that no poults fed wet mash and brooded in tiers developed deformity of the tongue. The only poult fed wet mash

TABLE III

INFLUENCE OF BREED ON THE INCIDENCE OF CURLED TONGUE IN AFFECTED GROUPS

	Standard bred Bronze	Standard bred × Broad-breasted Bronze	Broad-breasted Bronze (home bred)	Broad-breasted Bronze (purchased)	Broad-breasted White (purchased)	Beltsville Small White
Original No. of Poults ...	143	197	156	55	44	260
Percentage which developed Curled Tongue	32·8	20·8	5·8	9·1	Nil	6·9

which showed curled tongue had been brooded from day old on a solid floor.

The incidence of curled tongue among the poults of the four breeds and the cross-bred mating is shown in Table III. No breed, apart from the single hatch of purchased Broad-breasted Whites was free from the condition, but poults of the Standard-bred Bronze and of the cross involving this breed were much more seriously affected than either the Broad-breasted Bronze or the Beltsville Small White. This cannot be attributed to differences in the number of day-old poults used in the experiments, for of the home-bred stock more Beltsville Small White and fewer Standard-bred Bronze were involved than any other breed.

The reproductive performance of the Standard-bred Bronze was consistently inferior to that of the cross mating or of the other breeds and over the entire season the hatchability of initial eggs set was only 41 per cent. compared with 72 per cent. for one pen of Beltsville Small White. Further, the Standard-bred Bronze poults which hatched lacked vitality and this may have been a contributory factor in predisposing curling of the tongue on a dry mash diet.

Summary and Conclusions

A curled tongue condition in turkey poults characterised by a folding back of the distal end of the tongue on to the dorsal surface has previously been reported both in the U.S.A. and in Britain. The deformity appears during the early weeks of life and although both genetical and nutritional causes have been suggested, the precise aetiology has remained obscure.

In the present series of experiments poults of four different breeds and of one cross-bred mating developed typical symptoms of curled tongue when fed a home-compounded dry mash. The condition also occurred when a purchased turkey starter mash was used in a dry state. The incidence of the trouble in affected groups varied from 5·6 to 26·3 per cent. of the original number of poults.

When the diets were fed in a moistened condition from the time of hatching, only an isolated case of curled tongue was observed. A large proportion of affected poults recovered when a change was made from dry to wet mash feeding. The conclusions derived from this study are as follows :—

(1) The primary cause of a curled tongue condition in turkey poults is through feeding all mash diets of fine physical consistency in a dry state during the first few weeks of life.

(2) The visual appearance and the texture of a mash is an unreliable guide to its suitability for feeding in a dry condition to turkey chicks.

(3) The results of these and other trials indicate that when 70 per cent. or more of the weight of a mash comprises particles below 1 m.m. in diameter, it is liable to give rise to curled tongue when fed in a dry condition to young turkey poults. This value is likely to be subject to wide variation according to the nature of the ingredients included in the mash, the levels at which they are incorporated and, in particular, their predisposition to become sticky when moistened. As a precautionary measure it seems desirable to feed turkey poults during the first two or three weeks of life a diet of wet mash and/or crumbs or pellets.

(4) A very high rate of recovery from curled tongue occurs when the diet is changed from dry to wet mash.

(5) There is no evidence that inherent factors, the nature of the maternal diet or the chemical constitution of the turkey starter diet had any effect on the incidence of curled tongue.

Acknowledgments.—We are grateful to Mr. J. Getty for undertaking the physical examination of the mash, and to Mr. H. E. Roberts for assistance with histological work.

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DUBLIN GRADUATES REUNION DINNER

Arrangements have been made for a Dublin Graduates Reunion Dinner to be held in Thompson's Restaurant on Tuesday, September 6th, at 6 p.m. Tickets will be 14s. per head.

As it is extremely difficult to anticipate the number of graduates and friends wishing to attend this function, it would be appreciated if those interested would make application for their tickets (enclosing subscriptions) *before* Saturday, August 27th, so that final reservations and financial arrangements may be completed in good time. Applications should be addressed to:—

Mr. J. C. G. McCrea,
Hon. Sec. Organising Committee,
County Chambers,
Market Street,
Omagh,
Co. Tyrone,
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Accidental Nitrofurazone Poisoning in Baby Chicks

BY

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Introduction

SINCE about 1949 nitrofurazone (5-nitro-2-furaldehyde semicarbazone) incorporated in the feeding mash has been administered to poultry in the treatment and prevention of coccidiosis. It is known that above a certain concentration the drug is toxic, but the brief mention by Horton-Smith & Long (1952) seems to be the only record of toxicity in Great Britain. In America, Daily (1948) found that toxic doses in animals produced hyperexcitability of the central nervous system. Harwood & Stunz (1949a) found that a single dose of nitrofurazone given to 10 to 14-week-old male chicks, at the rate of 150 to 200 mg. per kg. bodyweight caused toxic symptoms which included depression in some chicks and hyperexcitability in others, occurring as early in some as four hours after feeding. The latter symptoms were manifested by rapid movements, loud squawking, extended rigid legs and wings, opisthotonos, and the chicks flinging themselves about as if decapitated. This exertion usually terminated fatally, the majority of deaths occurring on the second and third day after administration of the drug. In addition these authors (1949b) later found that when nitrofurazone was incorporated in the food of day-old chicks and fed continuously for 14 days at a level of 0·022 per cent. of the mash it caused some retardation in growth. A similar effect on growth was recorded by Gardiner & Farr (1954) when the drug was fed at this level for seven days, and by Peterson & Hymas (1950) who noted that 0·04 to 0·05 per cent. of the drug in the diet of four-week-old chicks caused retarded growth and deaths when fed continuously for nine days. The findings of Harwood & Stunz (1950) suggested that continuous feeding of the drug at a level of 0·0067 per cent. of the food to two-week-old chicks increased the frequency and severity of curled toe paralysis.

In turkey poults Moore & Brown (1950) observed that a single large dose of the drug caused ruffled feathers, inco-ordination of movement, spastic head and body movements, exhaustion and death.

This paper discusses the toxic effects that followed the inadvertent feeding of excess nitrofurazone to baby chicks.

History

Six groups of 150 day-old chicks were under experiment for the purpose of measuring weight gains on different diets. The groups were kept under identical conditions in separate runs in a single large building, littered on wood shavings and provided with adequate heat from infra-red or electric radiators.

The chicks came from two sources, X and Y (Table I).

1. *Source* X

These chicks were crossbred Light Sussex birds of both sexes. They had a prolonged rail journey in very cold weather and 10 out of 310 were dead on arrival. From the survivors two equal groups of 150 chicks each were made up (Groups 1 and 2).

Group 1 was fed a chick starting meal A in which nitrofurazone at a level of 0·011 per cent. had been incorporated during manufacture, and Group 2 was fed another chick starting meal B to which nitrofurazone was added before it was fed.

2. *Source Y*

These chicks were Light Sussex males and there were

TABLE I

DAILY RECORD OF DEATHS IN SIX GROUPS OF 150 DAY-OLD CHICKS, THREE OF WHICH (GROUPS 2, 4 AND 6) RECEIVED EXCESS NITROFURAZONE IN THE FOOD

Time after feeding (days)	Food A		Food B			
	Crossbred Light Sussex ♂, ♀ (Source X)		Light Sussex ♂ (Source Y)			
	Group 1 nitrofurazone	Group 2 nitrofurazone	Group 3	Group 4 nitrofurazone	Group 5 arsenic	Group 6 nitrofurazone and arsenic
1	—	15	—	3	1	7
2	—	11	—	2	—	1
3	1	20	—	10	—	16
4*	1	23	—	20	—	10
5	—	7	—	3	—	—
6	—	—	—	—	—	—
7	—	—	—	—	—	3
Totals	2	76	0	38	1	37
Nitrofurazone per cent. (analysis)	0·011	0·07	0·00	0·04	0·00	0·06

* Diet changed at the beginning of the fourth day.

no deaths on arrival. They were divided into four groups (Groups 3 to 6).

Group 3 was fed chick starting meal B without nitrofurazone ; Group 4 the same meal with nitrofurazone ; Group 5 was fed the same meal with arsenic as 3-nitro-4-hydroxy phenyl arsenic acid at a level of 0·005 per cent. but without nitrofurazone. Group 6 was fed the same diet as Group 5 but with the addition of nitrofurazone.

The chick starting meal B contained no nitrofurazone when purchased. In the case of Groups 2, 4 and 6 the drug was added to the food at an estimated level of 0·011 per cent. but owing to an error, which resulted in the pure drug being used instead of a premix, a much larger quantity was incorporated.

Observations on the Chicks

On arrival the chicks were divided into their respective groups and provided with food and water. At this stage all of them appeared normal. Twenty-four hours later 26 chicks were found dead (Table I), 15 from Group 2, three from Group 4, one from Group 5, and seven from Group 6. In addition most of the chicks in Groups 2, 4 and 6 were less active than those in the other groups and showed inappetence, while a few showed marked depression. A small number showed symptoms of hyperexcitability, manifested by cheeping in a high-pitched shrill note, dashing about madly amongst the other chicks, whirling in circles, flopping and jumping about like decapitated birds and finally dying in a state of collapse. Another unusual symptom shown by some chicks was the occasional stretching backwards of one of the legs in a way quite unlike leg stretching of normal birds. Chicks in the groups given nitrofurazone (2, 4 and 6) continued to exhibit these symptoms and on the third day chemical analysis of the food confirmed the suspicion that an error in dosage had been made. The diet of all groups was changed on the fourth day to one containing no nitrofurazone and after a further 48 hours no more deaths occurred except for three in Group 6 on the seventh day. By the end of a week after the commencement of the experiment a total of 154 out of 900 chicks had died, all except three of them being in Groups 2, 4 and 6, which were the only groups receiving excess nitrofurazone. The total mortality in the birds receiving excess nitrofurazone (Groups 2, 4 and 6) was 30 per cent. (151 of 450). The remainder recovered with change of food but the survivors were retarded in growth.

Post-mortem Examination

The only consistent macroscopic abnormalities observed were some oedema of the carcase, and venous congestion, especially of the lungs. The brain appeared macroscopically normal and sections stained with haematoxylin and eosin showed no obvious pathological lesions, either in the brain substance or the meninges.

Cultures were made from the heart blood, liver and lungs on to blood agar and desoxycholate citrate agar plates from 10 carcases from each of Groups 2, 4 and 6, and from the three birds that died in Groups 1 and 5. No significant organisms were isolated.

Chemical Analysis of the Food

Analysis of the original food given to all groups (Table I) revealed that this food given to Groups 2, 4 and 6 contained four to seven times as much nitrofurazone as had been intended (0·011 per cent.), and as was present in the diet of Group 1.

Discussion

These findings show that nitrofurazone incorporated in the food at a level four times that recommended for prophylaxis (0·011 per cent.) was toxic for baby chicks, which is in agreement with the findings of Peterson & Hymas (1950) for four-week-old chicks. On the other hand Horton-Smith & Long (1952) fed the drug at this level for seven days to two-week-old chicks and suggest that it was only slightly toxic or unpalatable. Neither group of workers observed nervous symptoms.

Some deaths occurred as early as 24 hours after feeding the drug but most deaths occurred after three days' continuous feeding. No further nitrofurazone was fed after the end of the third day by which time one-third of the chicks had died. It is possible that had the feeding of the drug at these levels been continued for several days longer many more of the chicks would have succumbed. The acute symptoms and heavy mortality after feeding for only three days suggest that the drug is relatively more toxic for day-old than for older chicks.

That the survivors were retarded in growth is in agreement with the observations of Harwood & Stunz (1949b), and Gardiner & Farr (1954). Horton-Smith & Long (1952) observed a temporary check in the growth of week-old chicks which they attributed to nitrofurazone feeding for 28 days at a level of 0·011 per cent. In our case also the retardation of growth was only temporary for by 10 weeks of age the birds were as well grown as those that did not receive excess nitrofurazone.

The nervous symptoms appear to be characteristic and closely agree with the observations of Harwood & Stunz (1949a) in 10 to 14-week-old chicks and Moore & Brown (1950) in turkey poults.

In contrast to Harwood & Stunz (1949a) who found catarrhal enteritis and marked congestion of the kidneys, meninges, and cranial bones at autopsy, we observed only general venous congestion and some oedema. Histological examination of the brain and meninges showed no abnormality, which agrees with the findings of Jungherr (1953). The early deaths which occurred in Group 2 without prior nervous symptoms, together with non-specific *post-mortem* and negative cultural findings and a history of a previous long and cold train journey at first suggested " chilling " as a possible cause.

The nervous symptoms are characteristic and in their absence it is likely that a diagnosis could be made only as a result of chemical analysis of the food.

Two groups (5 and 6) received arsenic in the diet but whereas 37 birds died in Group 6 which received excess nitrofurazone in addition to arsenic, only one bird died in Group 5 on arsenic alone. Arsenic is thus unlikely to have been the cause of toxicity.

Nitrofurazone poisoning in the field is unlikely to occur except through an error of dosage and it is unlikely that the two deaths that occurred in Group 1

(Concluded at foot of Col. 1 overleaf)

were associated with nitrofurazone for the drug was fed to this group at a level (0·011 per cent.) well recognised to be harmless.

Summary

Of 450 day-old chicks receiving excess nitrofurazone (0·04 to 0·07 per cent. estimated by subsequent analysis) in the diet for three days, 151 died and the survivors were retarded in growth. A characteristic symptom was hyperexcitability, with whirling in circles, ending in convulsive movements like those of a decapitated bird. No specific lesions were observed.

Acknowledgment.—I wish to thank Miss J. E. Dixon, A.R.I.C., of Messrs. Bibby's Nutrition Research Laboratories for analysis of the food for nitrofurazone.

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Current Literature

DIGEST

Enterotoxaemia (*Cl. welchii*) in calves

A report by Schofield suggests that sudden death in calves, one to three months old, can be caused by *Clostridium welchii* (Type A). According to this author, the "alpha" toxin is produced in the small intestine and, after absorption, affects the heart causing fibrillation. The *post-mortem* findings in affected animals were epicardial and endocardial haemorrhages, congestion of the duodenal and jejunal mucosa, pale and swollen livers, and pale and friable kidneys sometimes "pulpy" in consistency. Mice died immediately after being injected intravenously with the centrifuged supernatant fluid from the intestinal contents of suspected cases. This disease, which is confined to well-fed calves in prime condition, may be prevented by lowering the protein content of the feed, and vaccinating with a type A antitoxin, at three-weekly intervals from the age of one to three months.

Agalactia in sows

In answer to a query, Boley listed the common causes of agalactia in sows as metritis, mastitis, hypocalcaemia, and severe chilling during parturition. He also observed that some recently farrowed sows and gilts showed no symptoms except absence of milk, and for these cases he recommended posterior pituitary injections.

Forceps for the removal of foreign bodies

A pair of forceps which have been designed for the removal of foreign bodies in the upper digestive tract of dogs or cats are described by Antelyes. A metal tube, 23 inches in length and $\frac{1}{4}$ inch in diameter, contains a steel rod one end of which is divided into two arms, each of which is expanded to form jaws similar to that on a pair of small whelping forceps. The other end of the rod threads into the tube, and carries a handle. Rotation of this handle causes the jaws at the end of the rod to move forward and spread, while rotation of the handle in the opposite direction makes the jaws come together and retract into the tube. The forceps are passed by mouth into the oesophagus, and, if necessary, the stomach of an anaesthetised animal. Foreign bodies may be grasped by the jaws of the forceps through the appropriate manipulation of the handle, and withdrawn into the mouth.

Cases are described in which the forceps were used successfully to retrieve a stone and fish vertebrae from the thoracic oesophagus, and the partially decomposed body of an, apparently indigestible, rat from the stomach of a dog.

Human rabies in the U.S.A.

Official records quoted in *Veterinary Medicine*, **50** (3), give the total number of persons dying from rabies in the U.S.A. during the period 1943–51 as 276. This total includes 143 children under 14 years of age, who represent the most susceptible age group.

Diagnosis of ringworm

An outbreak of ringworm in a cat breeder's establishment is described by Steele in which *Microsporum canis* was demonstrated in 40 Persian cats and one dog by culture methods. However, only 60 per cent. of these animals had visible lesions, and only one-third exhibited fluorescence when exposed to Wood's lamp. The report continues with a disturbing account of a stray kitten, which was incriminated as the cause of ringworm among three women, eleven children, one baby and three dogs.

D. W. J.

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ABSTRACTS

Studies in Bracken Poisoning in Cattle. EVANS, C., EVANS, R., & HUGHES, L. E. (1954). *Brit. Vet. J.* **110.** 295–306, 365–380, 426–442.

The susceptibility of different species of farm animals to bracken poisoning is discussed in relation to their grazing habits. In cattle the problem is greatest on hill farms, but outbreaks are often sporadic depending on ecological conditions which cause scarcity of pasture herbage, or of actual roughage in an otherwise adequate herbage. Calves will also often eat bracken bedding.

In simple stomached animals, such as the rat and horse, bracken activity results in an avitaminosis B_1 due to thiaminase action on the vitamin. Because of rumen bacteria, this does not occur in cattle and the lesions are different, being mainly haemorrhagic in type. An "enteric" type in adults, and a "laryngitic" type in calves are described. High temperature (107° to 109° F.)

is a constant feature in the terminal stages. Both types differ from the inco-ordination syndrome in horses.

Bracken poisoning is known to have occurred eight weeks after removal from access to the plant. Morbidity is low, but mortality rate is high, and prognosis grave. No therapeutic agent of value has been found, but some animals may recover unaided.

Experimental work on yearlings and calves showed that bracken poisoning can be reproduced by feeding harvested green bracken and hay (50 per cent. each) over a period of approximately one to two months. Both clinical forms occurred.

Bracken poisoning occurred in bracken-fed animals receiving :—

(1) Dried yeast in the diet.

(2) Aneurin ; aneurin + nicotinamide ; or aneurin + B complex parenterally.

There was no therapeutic effect from :—

(1) Aneurin by mouth and parenterally.

(2) Aneurin by mouth and B complex members + vitamin C parenterally.

(3) Nicotinamide by mouth.

(4) Nicotinamide, aneurin, folic acid + "Plexan" (Glaxo) parenterally.

(5) B complex, + C, + "Plexan" parenterally.

Steaming bracken for about an hour prevented the disease occurring. In one experiment, mixing bracken with molasses prevented bracken poisoning, but it was subsequently found to have no effect.

Observations on the blood revealed (1) that severe leucopenia and thrombocytopenia occur before the onset of symptoms, and that these are related to an aplasia of the granulocytic and thrombocytic elements in bone marrow. To a lesser degree, and in the terminal stages, an erythrocytopenia with corresponding reduction of the erythrocytic precursors in the marrow occurs. This effect on the bone marrow is considered to be the fundamental lesion. Bone marrow was obtained for examination by sternal biopsy. (2) Capillary fragility is markedly increased ; there is defective clot retraction, and prolonged bleeding time. (3) The aneurin status in cattle suffering from bracken poisoning is not abnormally low, and differs from that in the horse and rat. There is a sharp terminal rise only of blood pyruvate.

A variety of bacteria was isolated by blood culture in the terminal stages. There is no relationship between positive blood cultures and fever.

Two field outbreaks of bracken poisoning in cattle are described, and the three sections of the paper are discussed and summarised.

During the summer of 1950 a bunch of heifers grazing hill pasture, where keep was short, became affected with bracken poisoning, developing nasal and anal haemorrhages and losing condition. Twenty-two died in all, the last death being in October, seven to eight weeks after direct access to bracken. None of the in-calf heifers running out with the others died. Detailed observations on four of these animals, and 12 in a subsequent outbreak on a different farm in 1951, confirmed the haematological and other findings in the experimental work. Various forms of therapy, including blood transfusion, were again found to be useless. It was found by haematological work that it was possible for some animals, not showing obvious symptoms, to recover slowly from the damage done to the bone marrow.

The pathology of bracken poisoning is discussed in relation to haemocytological depression.

Depression of bone marrow activity is the fundamental lesion. The origin of other characteristic symptoms of bracken poisoning are also considered and the difference between the aetiology in simple stomached animals and in ruminants is discussed. The presence of thiaminase in bracken accounts for the development of an avitaminosis B_1 in those animals which require a supply of aneurin in the diet. Although conditions in the rumen are suitable for thiaminase action, it is unlikely that the enzyme would attack intracellular aneurin produced by bacterial synthesis. In the ruminant no gross aneurin deficiency results, consequently haemocytological and osteo-myeloid depression are probably related to some other thermolabile or volatile factor in bracken, which is not one of the known vitamins or other substances which were given in therapeutic trials without effect. The substance might be modified in the rumen, or the product of interaction of active (but not steamed) bracken and rumen contents. The damage it does to the bone marrow is apparently irreversible after reaching a certain stage, after which recovery is impossible. Bracken poisoning is compared with other conditions in cattle producing similar symptoms and lesions, particularly tri-chlorethylene extracted soya bean oil meal poisoning.

C. M. E.

Vetafil—A Synthetic Suture Material New to North America. MILNE, F., & HORNEYS, F. (1954). *J. Amer. vet. Ass.* **125.** 448–449.

Vetafil is a white, synthetic material consisting of several strands coated with a special surface film. It may be sterilised by boiling or be supplied in sterile containers. It is more flexible than monofilament nylon and has a greater tensile strength than catgut. The authors have used Vetafil (0·6 mm.) for the repair of umbilical hernia in the calf and foal, and in bovine rumenotomies and caesarean sections. Vetafil (0·4 mm.) was used in ovine laparotomies. This material has been used to close both the abdominal wall and cutaneous incisions.

In Germany satisfactory results have been obtained with Vetafil. Merkt, however, has stated that it tended to cut through the uterine tissues when used in caesarean sections in cattle, sows and goats.

L. C. V.

Domestic Animal and Man in the Life Cycle of the Bovine Tubercle Bacteria. CHRISTIANSEN, M. J. (1953). 15th International Veterinary Congress. Part I. Vol. I. pp. 155–159.

Owing to the virtual eradication of tuberculosis in cattle in Denmark, Christiansen has been able to survey the position and establish that there exists a complete cycle from cattle to man and from man back to cattle, and, in addition, that a human being with bovine pulmonary tuberculosis could infect another human being with the bovine strain.

G. F. B.

The Victoria Veterinary Benevolent Fund

58th ANNUAL GENERAL MEETING

The 58th Annual General Meeting of the Victoria Veterinary Benevolent Fund (the 37th Annual General Meeting since incorporation) was held at 10, Red Lion Square, London, W.C.1, on June 1st.

There were present : Professor L. P. Pugh (Vice-President) in the chair, Professor T. J. Bosworth, Major J. J. Dunlop, Professor C. Formston, Mrs. D. I. Glover, Miss J. O. Joshua, Professors J. McCunn, W. M. Mitchell, Dr. R. F. Montgomerie, Messrs. J. N. Ritchie, J. B. White and Professor G. H. Wooldridge.

Mr. C. W. Francis, Secretary, was in attendance.

Apology for Absence

The Secretary (Mr. Cyril W. Francis) : I have a telegram from the President, Mr. H. W. Dawes, who regrets he is unable to travel to London due to the railway strike. Mrs. D. McNabb (President, Ladies' Guild) and Captain A. Whicher also send apologies for absence.

The Chairman : I am sure you will be sorry that Mr. Dawes is unable to get here due to inability to get transport in time.

Minutes

The Minutes of the previous Annual General Meeting held on June 1st, 1954, having been published were taken as read, and signed as correct.

Election of Officers

On the proposal of the Chairman, Mr. H. W. Dawes was re-elected President.

Mr. G. P. Male, Captain T. M. Mitchell, Professor L. P. Pugh and Professor G. H. Wooldridge were re-elected Vice-Presidents.

Major J. J. Dunlop was re-elected Hon. Secretary, and Professor J. McCunn was re-elected Hon. Treasurer.

The Auditors, Messrs. Wilkinson, Chater & Co., Chartered Accountants, 28, Queen Street, London, E.C.4, were re-appointed.

Annual Report and Statement of Accounts

The Chairman : You have all received this Report. Shall we consider it as a whole or take it page by page ?

Professor Mitchell : I think we usually take it page by page.

This method was adopted and the following discussion took place.

The Chairman : Page 6.

Professor G. H. Wooldridge : I think there should be some reference made to the Ladies' Guild and the money that has come in from them, as indicated on that page. I propose we should pass a very special vote of thanks to the ladies for the very great work they have continued to do for the Fund. At the same time I would like to include in our thanks those various other bodies who have worked so hard, in this country, in Wales, Scotland and Ireland.

Professor G. H. Wooldridge : Pages 11 to 14 : is there any additional information you would like to give to the members about the cases relieved ?

The Chairman : Nothing in particular. They are about the same in number. Every effort is made to increase the grants as far as possible, to meet the increased cost of living.

Page 15 : You will see that a new method has been adopted in presenting the accounts and the schedule of General Fund investments, which I think you will all agree is a great improvement. *Agreed.*

Professor Mitchell : How is this Fund building up ?

The Treasurer : It is gradually increasing from year to year. It is best not to stress this fact for it might have an effect on current subscriptions. Anyone who reads this Report can see the position of the Fund. We want to keep current subscriptions going, as well as building up our funds.

Professor Mitchell : The Treasurer should give us a reasonable general account of the Fund. There is not time, in a busy practice and a teaching post, to study all these reports, and he has the whole thing under his hand.

Professor Wooldridge : When you are making an appeal for charitable contributions, it never does to tell the people you are approaching that you have unlimited resources, or they react in the opposite direction.

The Chairman : Nothing was being withheld from the members, the financial state is set forth clearly in the report.

The Chairman moved the adoption of the Report, Professor Mitchell seconded.

The Annual Report and Statement of Accounts were adopted.

Election of 10 Members of the Association to Serve on the Council

The following were elected to the Council as representing the Members of the Fund : Mrs. D. I. Glover, Professor T. G. Browne, Dr. W. R. Wooldridge, Mr. G. N. Gould, Professor R. Lovell, Captain A. Whicher, Mr. E. P. Barrett, Professor W. L. Weipers, Messrs. A. Spicer and J. N. Ritchie.

In reply to Professor Mitchell, the Treasurer said the matter of re-election of representatives from the Divisions of the British Veterinary Association might come up automatically at the Divisional annual meetings.

It was pointed out that the Society of Women Veterinary Surgeons should have a representative, and that the Southern Counties also had no representative.

It was agreed that the Secretary should write to the Divisions concerned on this matter.

Any Other Business

Professor Mitchell proposed a vote of thanks to the Chairman, which was carried with acclamation.

University News

Cambridge

An impressive ceremony took place at Cambridge on Saturday, June 25th, in the Council Room of the University to mark the admission to the Royal College of Veterinary Surgeons of the first group of students to qualify for the new Cambridge degree of Bachelor of Veterinary Medicine, the courses for which started six years ago.

The Rt. Hon. H. U. Willink, Vice-Chancellor of the University, presided and was supported by Professors W. I. B. Beveridge and L. P. Pugh, while the Royal College was represented by Dr. W. R. Wooldridge and the Registrar. There was a large attendance of University teaching staff and relatives and friends of the new graduates.

The Vice-Chancellor, in welcoming Dr. Wooldridge, said that the University felt honoured in that the Royal College, in the unavoidable absence of the President, had sent its immediate Past-President to admit to membership the first recipients of the new Vet.M.B. degree. He himself had had the privilege the previous day of conferring the first of these degrees.

Dr. Wooldridge recalled that he had been a member of the Loveday Committee which had recommended the institution of the veterinary courses at Cambridge leading to the new degree and said that, though the beginnings might seem small, much was expected of the new School of Veterinary Medicine. Its students would enjoy the unrivalled advantages of close association with the University and would have the benefit of fine new buildings and equipment. It was to be hoped, therefore, that the graduates of this University would make a notable contribution towards the progress of veterinary medicine to higher levels in research and practice. Dr. Wooldridge finally drew attention to the declarations which had to be made on admission to the Royal College and recommended that members of the profession should always keep before them the ideal of service to the community.

The new graduates (whose names appear below) were then severally introduced to Dr. Wooldridge by Professor Pugh and signed the Roll of Membership of the Royal College of Veterinary Surgeons.

The graduates and others who had attended the ceremony were later entertained by Professor and Mrs. Pugh at a very pleasant sherry party at Magdalene College.

Veterinary Examinations

The following have passed the Second Part of the veterinary examination and have been admitted to the degree of Vet.M.B. :—

Mr. A. R. Akester, M.A. (St. Catherine's College)
Mr. I. G. Gibson, B.A. (Downing College)
Mr. M. D. N. Hunt, B.A. (Christ's College)
Miss F. Kenchington, B.A. (Newnham College)
Miss U. Parsons, M.A., PH.D. (Girton College)
Mr. E. M. Scratton, B.A. (Trinity Hall)
Mr. G. White, B.A. (St. John's College).

The following have now completed the First Part of the veterinary examination by passing in animal pathology and bacteriology, having previously passed the two other sections of this examination :—

Mr. M. R. Brambell, B.A. (Christ's College), and
Mr. D. L. Kingsberry, B.A. (Magdalene College).

Liverpool

B.V.SC. FIRST EXAMINATION

Part I: Chemistry, Part II: Physics, Part III: Botany and Part IV: Zoology

Aspinall, Kathleen M.; Barnes, J. D. F.; Batty, A. F.; Bird, Christine M.; Butterfield, W. E.; Callender, D. E.; Capron, T. K.; Chesworth, A. T.; Edwards, J. D. D.; Fitton, J.; Foot, J. K.; Hanford, D.M.; Holt, H. J.; James,W.; Jenkins, J. T.; McMurtry, M. J.; Michael, H. P.; Miller, J. K.; Nyavor, A.; Pickup, Pamela; Pinnel, J. A.; Quartey-Cofie, J. E.; Schofield, J. B.; Walker, J. M.; Weller, R. J.; Wild, G. S. P.; Worthington, P. T.; Zanetti, P. J.

Passed in individual subjects:

Adams, J. (Chemistry and Physics); Fritton, M. W. (Chemistry, Physics and Zoology); Goulden, M. B. (Chemistry, Physics and Zoology); Pickering, P. L. (Chemistry, Physics and Zoology).

B.V.SC. SECOND EXAMINATION, JUNE 1955

Part I: Veterinary Anatomy (including Histology and Embryology)

Francis, P. G.; Hurst, D. S.

Part II: Physiology (including Biochemistry)

Barr, D. A.; Death, R. W. J.; Mears, Hazel; Waterworth, J.

B.V.SC. THIRD EXAMINATION

Part I: Pharmacology

Barrett, S.; Bolger, K.; Butler, C. L.; Crabb, P. J.; Davies, E. B.; Dickinson, L. G.; Ewbank, R.; Fraser, C. E. O.; Harrison, F. A.; Hobson, G. J.; Holmes, J. A. R.; Howell, J. M.; Hughes, I. B.; Jenkinson, T. A.; Knifton, A.; Lancaster, R.; Menear, H. C.; Murgatroyd, A. E.; Pickard, Barbara; Tandy, J.; Tyler, J.; Whiteley, H.

B.V.SC. FINAL EXAMINATION, JUNE, 1955

Adams, D. V. N.; Bate, T. C. (with distinction in Regional Veterinary Surgery and Obstetrics); Boyd, R. W.; Caldwell, B. A. W.; Chandler, N.; Clarkson, M. J.; Copley, J. D.; Evans, R. K.; Farrer, J. S.; Featherstone, P.; Glaister, A. I. E.; Gleave, P.; Greenwood, J.; Hudson, G.; Potts, R. R.; Rothwell, J.; Sanford, J.; Scambler, J. D.; Smith, M. W.; Wallis, A. S.; Walters, T. M. H.; Wheeler, A. J.; Wright, C. L.

Passed in individual subjects:

Blair, Ruth Joyce (Veterinary Surgery); Shepherd, P. R. (Veterinary Medicine).

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At a ceremony to be held on Saturday, July 2nd, the degree of M.V.Sc. will be conferred on Mrs. E. J. Castle (*née* Bailey), B.V.SC., M.R.C.V.S., who has been working in the Department of Veterinary Preventive Medicine, and on Miss N. Wickham, B.V.SC. (Sydney), lecturer in Veterinary Pathology.

The title of the thesis presented for the degree by Mrs. Castle was "Factors affecting the rate of passage of foodstuffs in the goat," and the title of the thesis presented by Miss Wickham was "The haemagglutinin of *Clostridium welchii*."

News and Comment

VISIT OF CANADIAN MINISTER OF AGRICULTURE

The Canadian Minister of Agriculture, the Rt. Hon. J. G. Gardiner, will arrive in this country on Saturday, July 2nd, for a visit as guest of Her Majesty's Government. Mr. Gardiner, who was previously Premier of Saskatchewan, became Minister of Agriculture for Canada in 1935. Since then he has served continuously as Minister of Agriculture. In addition, for a period in 1940 and 1941 he was also Minister of National War Services. He was appointed Privy Councillor in 1947.

He will be including a number of visits in his engagements here. Notable among these will be a visit to the veterinary laboratory of the Ministry of Agriculture, Fisheries and Food at Weybridge. He will also see arable and mixed farming in Hertfordshire; fruit and dairy farming in Kent; and beef cattle, arable and hill farms in Scotland.

DIRECTOR OF ZOOLOGICAL SOCIETY ACQUITTED ON CRUELTY CHARGE

A case was tried at the Edinburgh Sheriff Court recently in which Mr. Donald Bowles, Director-Secretary of the Royal Zoological Society of Scotland, was charged with cruelty to horses at the Corstorphine Zoo, Edinburgh.

In finding Mr. Bowles not guilty, the presiding Sheriff said that he reached that decision not on mere technical grounds but on the merits of the case.

It was alleged that in January this year the defendant caused unnecessary suffering to five horses belonging to the Zoological Society by causing them to be kept in an open field without shelter. In giving evidence, Mr. J. W. Burgess, M.R.C.V.S., senior lecturer in zoology at the Royal (Dick) School of Veterinary Studies, said that if the horses' winter coat was grown, there was no unnecessary suffering in keeping horses out of doors in winter conditions.

Lord Rosebery also in evidence said he had complete confidence in Mr. Bowles's care and skill. He added that he himself had kept his hunters in open fields and found that they benefited from the cold weather when they could be in the fresh air without being worried unduly by flies and insects.

The President of the British Veterinary Association, Professor Robertson, gave evidence also and said that it was common practice to winter Clydesdale horses out of doors. This did not cause unnecessary suffering under conditions in Scotland.

Evidence for the prosecution was given by officials of the Scottish Society for the Prevention of Cruelty to Animals, but the decision for the defendant was as recorded above.

HAMPSHIRE CATTLE BREEDERS' SOCIETY

We have received a copy of the Annual Report for 1954 of the Hampshire Cattle Breeders' Society. The organisation has completed another year of great activity under the chairmanship of Mr. G. N. Gould who, as readers will know, is chairman of the Technical Development Committee of the B.V.A. and also a prominent member of the Council of the Royal College of Veterinary Surgeons. In the course of his speech Mr. Gould said:

"This year we have reached a stage in the affairs of the Hampshire Cattle Breeders' Society that seemed but a dream a few years ago.

"I can now report that the whole of the initial loan capital has been paid off, that we have purchased a site at Lyndhurst to build a new centre to give full and adequate accommodation for our growing activities, and further that we have the financial resources available to build such a centre—the plans for which are in process of discussion by the Committee.

" This, however, is not the end of the story. The number of first inseminations increased in 1953 by 7,529, in 1954 by 6,570 and, this year, there is an increase in comparative figures of more than 25 per cent. over the 1954 record numbers, indicating that we are likely to inseminate about 50,000 cows and heifers this year.

"Continued progress has been made with the field use of deep freeze semen and the selection of semen which will give satisfactory fertility with this method of storage.

"Statistical analysis of what is probably the largest field trial of its kind in the world—covering more than 30,000 first inseminations over a period of two years, three months, at a conception rate of 60 to 62 per cent., is now being made by Mr. Clifford Thomas of Southampton University. Valuable information concerning the evaluation of semen may be extracted from the technical data involved. Many deep freeze calves have been born—the first to one of Mr. Gosling's heifers at New Park in November, 1953. Some have been served by deep freeze semen.

"We have also commenced a field trial of insemination of pigs, using a landrace boar. Twenty-five sows and gilts have been inseminated to date and it is being hoped to complete the initial trial of 100 in the next few weeks."

JOURNAL OF THE MALAYAN V.M.A.

We have been pleased to receive recently the first issue of the journal of the Malayan Veterinary Medical Association. The Association was formed in 1939, but owing to the war and its aftermath, and to other problems, it has not been found possible to launch this journal until now. The first issue is pleasantly printed on good quality paper and contains several clinical and scientific articles by local veterinarians. There are also abstracts from current literature and some notes on the work of the Association.

In view of the fact that there are only about 75 members of the Malayan V.M.A., the production of this journal is a courageous effort upon which its originators should be congratulated.

Veterinary surgeons and research workers in other countries are invited to contribute to its columns, and there may be those among our readers who may feel that they would like to give a helping hand to this new periodical.

MEETING ON SHEEP DISEASES

There was an attendance of nearly 400 at a meeting on sheep diseases organised by the Animal Health Division, Ministry of Agriculture, Fisheries and Food, at Malton.

Professor Beveridge of the Department of Animal Pathology, Cambridge, spoke on Infectious Diseases of Sheep, paying especial attention to foot rot. He showed a film, prepared in Australia, which illustrated the principles involved in eradicating this disease and in his talk and discussion he stressed the fact that eradication of foot rot from flocks in this country could be achieved. Various other diseases including enterotoxaemia, contagious pustular dermatitis, and virus and bacterial abortion were also covered in his talk and the ensuing discussion.

Dr. E. L. Taylor, C.B.E., of Weybridge, gave the second paper on Parasitic Diseases of Sheep. Dr. Taylor stressed the fact that successful management of sheep implied the successful management of parasitic worms which occurred wherever sheep were kept. Good farmers practised rule-of-thumb methods that enabled them to control parasites, and he explained how these practises brought about these beneficial effects. He illustrated his talk with a series of slides and during the discussion answered many questions on the control and treatment of parasites in relation to various methods of farming.

Professor J. McGregor, of the Department of Agriculture, Leeds University, introduced the speakers to the audience and in his summing up expressed the view that if farmers made use of the knowledge they had gained from the two papers, unnecessary wastage of sheep could be avoided.

RETIREMENT OF MR. R. S. MARSHALL FROM NIGERIA

The Colonial Office have recently announced the retirement of Mr. Robert S. Marshall, C.B.E., M.R.C.V.S., D.V.S.M., from the post of Director of Veterinary Research, Nigeria, and his appointment as Adviser on Animal Health to the Secretary of State for the Colonies. Mr. Marshall succeeds Mr. R. J. Simmons, C.M.G., C.B.E., to the latter post.

Mr. Marshall's retirement marks the end of an active and varied career in the Colonial Veterinary Service. After his early education at Hamilton Academy he obtained the National Diplomas in Agriculture and in Dairying at the West of Scotland Agricultural College in 1923 and qualified as M.R.C.V.S., Glasgow, in 1926. He took his D.V.S.M., Edinburgh, in 1927, and Diploma in Bacteriology, London, in 1929, being one of the first veterinarians to obtain the latter diploma.

Mr. Marshall's first appointment to Nigeria was as Veterinary Pathologist in the then newly formed Department of Trypanosome Research, where he carried out valuable pioneer research work. In 1934 he joined the Veterinary Department, Nigeria, becoming Senior Veterinary Research Officer in 1938, Principal of the Veterinary School, Vom, in September, 1944, and Deputy Director of Veterinary Services in December, 1948.

Under the new constitution Mr. Marshall became the first Inspector-General of Animal Health Service, Nigeria, in October, 1951.

More recently, as the regions became completely autonomous under the revised constitution, his rôle changed from Inspector-General, Nigeria, to that of Director of Research and Adviser to the Federal Government.

Mr. Marshall says "good-bye" to the Hon. Peter Achimugu, Minister of Natural Resources, N. Region, Nigeria.

Mr. Marshall's retirement from Nigeria marks the end of what might be termed an epoch, associated especially with the names of past departmental leaders, Captain W. W. Henderson, Mr. Griffiths, Mr. R. J. Simmons, Mr. W. G. Beaton and notable for the rapid advances made in the field control of epizootic diseases, the increased use of biologicals in the control of the more important economic sicknesses of livestock and the spectacular growth of a very lucrative animal industry. In all these developments Mr. Marshall played an active part, his wide knowledge and experience being of great value during this critical period of expansion.

MINISTRY OF AGRICULTURE VETERINARY FIELD STAFF CHANGES

Mr. F. H. Addison, B.SC., M.R.C.V.S., D.V.S.M., Divisional Veterinary Officer at Head Office, Tolworth, has been transferred to Exeter, and Mr. J. C. Baird, B.V.SC., M.R.C.V.S., Divisional Veterinary Officer at Liverpool Quarantine Station, has been transferred to Mold. Mr. E. T. Camps, M.R.C.V.S., Veterinary Officer at Yeovil, has been transferred to Head Office, Tolworth, on promotion to Divisional Veterinary Officer. Mr. J. G. Crowhurst, M.R.C.V.S., Veterinary Officer at Leicester, has been transferred to Liverpool Quarantine Station as Divisional Veterinary Officer on promotion. Mr. W. S. MacKay, M.R.C.V.S., Divisional Veterinary Officer at Carmarthen, has been transferred to Taunton. Mr. R. B. T. Munro, M.R.C.V.S., Veterinary Officer, has been promoted to Divisional Veterinary Officer and remains at Preston. Mr. J. W. Simpson, B.SC., M.R.C.V.S., Divisional Veterinary Officer at Mold. has been transferred to Carmarthen, while Mr. J. M. Ware, B.SC., M.R.C.V.S., Veterinary Officer at Shrewsbury, has been transferred to Chester as Divisional Veterinary Officer on promotion.

R.C.V.S. LIBRARY ACCESSIONS

CANADA. *Minister of Agriculture*. Report for the year ended March 31st, 1954.

FIRST CONGRESS OF THE FEDERATION OF YUGOSLAV VETERINARY ASSOCIATIONS. *Zagreb*. Zbornik. 1. Kongresa saveza drustava veterinara F.N.R. Jugoslavije, u Zagreb, 3-6, XII, 1953. [Collected reports] Zagreb.

ELDER, C., *and* RODABAUGH, D. E. Role of protein in immunization of swine against cholera. Columbia, Missouri A.E.S. 1954.

GREAT BRITAIN. Ministry of Agriculture and Fisheries. British poisonous plants, A. A. Forsyth. London, H.M.S.O., 1954.

GUYER, P. Q., *and* DYER, A. J. Study of factors affecting sheep production. Columbia, Missouri A.E.S., 1954.

HEDIGER, H. Studies of the behaviour of captive animals in zoos and circuses. London, Butterworth, 1955.

HOOVER, C. R., *and* TURNER, C. W. *In Vitro* metabolism of the rat mammary gland *and* Observations on *In Vitro* actions of thyroid hormones. Columbia, Missouri A.E.S., 1954.

MANN, T. The biochemistry of semen, London, Methuen, 1954.

NEW ZEALAND. *Veterinary Services Council*. Eighth annual report for the year ended March 31st, 1955.

TANGANYIKA. *Veterinary Department*. Annual report, 1954.

The librarian would be glad to contact anyone with any of the following books to dispose of (by gift or sale):—

BARTON, F. T. Cattle, sheep and pigs, London, Jarrold, 1920.

WALSH, J. H. ("Stonehenge" *pseud.*) The dogs of the British Islands. *Second edition*. London, Cox, 1872.

WALSH, J. H. ("Stonehenge" *pseud.*) The dog in health and disease. London, Longman, 1872.

PERSONAL

Births

BARNES.—On June 15th, 1955, to Judith (*née* Roddick), wife of E. G. Barnes, M.R.C.V.S., of Dereham, Norfolk, a daughter, Lucinda Judith.

BENNET.—On June 23rd, 1955, to Daphne, wife of J. R. Bennet, M.R.C.V.S., High Street, Helmsley, York, twin children—a boy and a girl.

ROBINSON.—On June 24th, 1955, to Jill, wife of Peter Robinson, B.V.SC., M.R.C.V.S., Egerton Hall, Malpas, Cheshire, a daughter, Janet Gaynor.

SMITH.—On June 14th, 1955, at Duke of York House, Bradford, to Pat (Audrey), wife of D. C. Smith, M.R.C.V.S., 431 Bradford Road, Thornbury, Bradford, a daughter, sister for Susan and David.

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

HART, Andrew (Capt., late R.A.V.C.), of The Anchorage, Ardnadam, Argyllshire. Graduated Glasgow, December 18th, 1894. Died June 23rd, 1955, aged 84 years.

MR. WILFRED RHODES

Mr. S. V. Collard writes:—

It was with great sorrow that we heard of the sudden death of Wilfred Rhodes, and it was a sad day on Wednesday, June 22nd, when his many friends and colleagues attended his funeral at Ipswich Crematorium. He had collapsed on the previous Saturday whilst playing golf, and died on Monday.

Wilfred Rhodes qualified in London in 1925 and also gained his B.Sc. degree. After seeing practice he was appointed as Veterinary Officer under the Surrey County Council, which appointment he held until the amalgamation with the Ministry of Agriculture in 1938. He then came to Warwick as Veterinary Officer until 1943 when he was promoted Divisional Veterinary Officer for East Suffolk and held that position until transferred to Head Office, where he was stationed at the time of his death.

I had known Wilfred for 34 years. At the London College and later at Warwick I was in close association with him. He was a good friend and colleague, of cheerful disposition and had the happy capability of extracting all the pleasures that life has to offer; it is tragic that his life has been terminated at the age of 53. He was a very good golfer and would no doubt have excelled himself in the top class if he had had more time to play. He was a modest winner and a good loser. He left a large circle of friends at all the towns in which he had lived and he did not forget them.

As we left Ipswich on Wednesday, it was with sad hearts that we said, "Good-bye, old friend."

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The funeral service, held at Ipswich Crematorium on June 22nd, was attended by many friends and agriculturists in East Suffolk, where Mr. Rhodes was Divisional Veterinary Officer until 1952.

Members of the profession who attended the service were: Mr. B. Claxton, representing the Chief Veterinary Officer and colleagues at Head Office, Tolworth; Mr. J. McLauchlan, representing the Association of State Veterinary Officers; Mr. D. Cameron, Mr. J. J. Gildea, Mr. T. Duffy, Major W. H. Wortley, Mr. J. MacQueen Brown, Mr. S. V. Collard, Mr. W. Nightingale and Mr. J. Brennan.

MR. A. H. MILNE

Mr. J. Newhouse writes:—

Those members who were at Edinburgh between the years 1933 and 1938 will remember Sandy Milne.

Not only was he a student who mastered his professional subject with ease, but his interests embraced a much wider sphere. In the field of athletics he was a member of the rugger team and for many seasons, the mainstay of the cricket club. As Editor of the "Centaur" he brought skill and energy to the task. His oratory was Churchillian and his talents in this respect were often called upon at College functions.

On qualifying he spent a short time in practice in Cornwall before joining the Colonial Service in Tanganyika. He published many of his works, some of which appeared recently in THE RECORD.

It was whilst travelling to Abyssinia with a view to ascertaining the possibilities of establishing a Veterinary College there, on behalf of the United Nations Organisation, that he met his death in the tragic plane disaster of May 20th.

His untimely end has robbed the Colonial Service and the profession of a most promising member. He leaves behind a widow, to whom we extend our deepest sympathy.

COMING EVENTS

July

6TH (WED.). Annual General Meeting of the A.V.T. and R.W.'s (Northern Region) at Harper Adams Agricultural College, Newport, Salop, 10.30 a.m.

14TH (THURS.). Summer Meeting of the Central Division, B.V.A., at The Royal Veterinary College, Streatley, Berks, 1 p.m.

20TH (WED.). Annual General Meeting of the Southern Region of the A.V.T. & R.W.'s at the Agricultural Research Council Field Station, Compton, Berks.

22ND (FRI.). Meeting of the Yorkshire Division, B.V.A., at Government Buildings, Crosby Road, Northallerton, 2.30 p.m.

28TH (THURS.). President's Invitation Luncheon and General Meeting of the Midland Counties Division, B.V.A., at the Swan Hotel, Stafford, 12.30 p.m.

September

4th to 10th. 73rd Annual General Meeting and Congress of the British Veterinary Association at Belfast.

B.V.A. COUNCIL AND COMMITTEE MEETINGS IN LONDON

Wednesday, July 6th.—

10.30 a.m. Home Appointments Committee, at 7, Mansfield Street, W.1.
12 noon Parliamentary and Public Relations Committee, at 7, Mansfield Street, W.1.
2.00 p.m. Organising Committee, at 7, Mansfield Street, W.1.

Thursday, July 7th.—

10.30 a.m. Veterinary State Medicine Committee, at 7, Mansfield Street, W.1.
2.30 p.m. General Purposes and Finance Committee, at 7, Mansfield Street, W.1.

Friday, July 8th.—

10.30 a.m. Council Meeting at the Connaught Rooms, Great Queen Street, Kingsway, W.C.2.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Dumfries. Newmains, Keir, Thornhill (June 21).

Durham. Copley Farm, Butterknowle, Bishop Auckland (June 23).

Kirkcudbright. Auchengibbert, Crocketford (June 21).

Northumberland. West Petrel Field Farm, Hexham (June 22).

Yorks. Grey Towers, Nunthorpe, Middlesbrough; Carlton Grove Farm, Carlton-in-Cleveland, Middlesbrough (June 20).

Atrophic Rhinitis

Herts. Old School Farm, Broxbourne (June 18).

Swine Fever

Berks. Henley Farm, Wantage Road, West Shefford, Newbury (June 25).

Bucks. Pyghtle Cottage, Village Road, Denham, Uxbridge (June 21). 80, Bradwell Road, New Bradwell, Wolverton (June 27).

Ches. Big Hind Heath Farm, Hind Heath Lane, Wheelock, Sandbach; Hame Farm, Moss Lane, Lostock Graham, Northwich (June 23); Oak Farm, Hoole Village, Chester; Whitehall Farm, Warmingham, Sandbach (June 27).

Denbigh. Tyn Celyn, Llanelian, Colwyn Bay (June 27).

Derbys. Highfield Hall Farm, Sheffield Road, Chesterfield; Carter Hall Farm, Ridgeway, Eckington (June 23).

Devon. Whimple House Farm, Whimple, Exeter (June 22).

Dorset. Cadmans, Marnhull, Sturminster Newton (June 21).

Essex. Sheering Hall Farm, Sheering, Harlow (June 24).

Flints. Tyddyn, Mord (June 22); Coppa Farm, Daisy Hill, Buckley (June 24).

Lancs. Glenside, Long Heys Lane, Dalton, Wigan; Holme Farm, Calder Vale, Barnacre, Garstang; Sowerby Lodge Poultry Farm, Barrow-in-Furness; Corner Farm, Great Plumpton, Kirkham (June 27).

Leics. The Rookery, Dunton Bassett, Rugby (June 27).

Norfolk. Dover Farm, Briston, Merton Constable (June 24).

Oxford. Ardley Road Farm, Bucknell, Bicester (June 21).

Staffs. 58, Park Road, Alrewas, Burton-on-Trent (June 22); 412, Blurton Road, Stoke-on-Trent (June 24); Baldwins Gate Farm, Baldwins Gate, Newcastle, Stoke-on-Trent (June 25); 17, Linehouses, Golden Hill, Stoke-on-Trent (June 27).

Surrey. Elm Farm, Farleigh, Warlingham (June 23).

Warwicks. Sties at Slade Lane, Hall Green, Birmingham (June 20); Woodside Farm, Duttons Lane, Sutton Coldfield, Birmingham; Brooke House Farm, Peterbrooke Road, Shirley, Birmingham; Westfield Farm, Barmby Moor (June 21); Northfield Farm, Staircase Lane, Allesley, Coventry (June 22); Benton Green Farm, Berkswell, Coventry; Truemans Heath Farm, Shirley, Birmingham; Barton Farm, Alderminster, Stratford-on-Avon (June 24).

Yorks. Fold Cottage, Goosehill, Warmfield, Wakefield; Denwell Allotments, Back Northgate, Pontefract; Coldstone House Farm, Sinderby, Thirsk (June 24); Hill Top Farm, Rooms Lane, Morley (June 27).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

"A WELL OF ENGLISH UNDEFILED"

Sir,—With such folk as Paul Crosfield and David Ogilvie waiting round the corner like, it takes a brave man to send anything to your paper nowadays. But they do say round here that if a fellow has anything to say worth hearing, other chaps will understand all right even if they come from next county.

Let them stop writing that have nothing to say, and a good riddance, and not to go making other people too nervous. I remember a poem in a book of my grandad's, by Mrs. Edward Craster. Went like this:—

"The centipede was happy quite
Until a toad in fun
Said, 'Pray which leg goes after which?'
That worked her mind to such a pitch,
She lay distracted in a ditch
Considering how to run."

Yours faithfully,

Lan House, ESMOND BROWN,
Weston Road,
Bath.

June 27th, 1955.

THE VETERINARY CLUB SYSTEM OF NEW ZEALAND

Sir,—As a veterinary surgeon who has spent nearly eight years under this system, I feel a few notes might be helpful to anyone who may consider taking up an appointment.

The scheme was fostered by groups of farmers who realised that professional guidance and assistance were required. On the other hand a number of farmers have openly stated that veterinary surgeons in New Zealand—private practitioners—would only do certain work and would not serve the farming industry as a whole. This was said only about ten years ago when there were something like 20 practitioners to cover an area larger than Great Britain. This idea is dying hard.

The result is seen in the club system in which the veterinary surgeon is paid a fixed salary for an unlimited amount of work, while the farmer pays a low subscription and also a ridiculously low fee for actual services rendered.

On more than one occasion a farmer has said: "I have paid my subscription and I demand your services." This is surely not a healthy veterinary surgeon-client relationship?

The farmer does not pay according to the nature of the work done, the time of day or the time spent on the premises. Nor does he pay according to the number of cases seen on the same farm at one visit. To quote Professor Cameron, of Canada, when he visited New Zealand: "An admirable scheme for the farmer, but not for the veterinary surgeon."

In the contract "approved" by the New Zealand Veterinary Association, about which I shall say little, there is a clause "extra pay for extra work." This clause has not been accurately defined, and it is only by the grace and goodwill of the club executive, approved by the Veterinary Services Council, that any extra remuneration can be obtained.

The salary is fixed by the Veterinary Services Council and there is no scale or foreseeable rate of remuneration after the "five years from graduation" scale. Here again it depends on whether the individual is in or out of favour, or whether the club finances will bear any increase.

The private practitioner suffers unfair competition from the clubs, in that the latter are subsidised by public and semi-public funds. The Veterinary Services Council are only prepared to assist private practitioners in very limited ways, and equally limited numbers.

In previous correspondence on the subject it has been said that the farmer does not control the veterinary surgeon. Perhaps someone will explain why the Council is not prepared to grant an equal voting strength to farmers and veterinary surgeons alike, instead of two farmer votes to one veterinary vote.

Our profession is an honourable calling and should be allowed to take its rightful place in the community. The farmer is master of his ship and captain of his crew. If he would allow the veterinary surgeon to attain a similar status, then each side could meet on a man for man basis and a very successful outcome would be the result.

The Veterinary Club scheme has done a useful task, but it is now time for a revision of the whole scheme. Otherwise, everything will be set for a nationalised veterinary service. The whole question is a matter of ethics and of mutual understanding rather than one of finance.

In case anyone is in doubt about the private practitioner in New Zealand may I say that, as always, he has given, and still is giving a first class service which is greatly appreciated.

Yours faithfully,

G. T. WILLIAMSON.

"Birchcroft,"
Sandhurst Road,
Wokingham, Berks.

June 13th, 1955.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period		Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
16th to 31st May, 1955		26	—	—	17	—	101
Corresponding period in	1954	10	1	—	13	—	91
	1953	17	—	—	30	—	201
	1952	25	—	101	3	—	44
1st January to 31st May, 1955		402	—	6	242	—	671
Corresponding period in	1954	136	1	12	512	—	643
	1953	395	—	25	277	—	1,470
	1952	243	—	302	109	1	499

TUBERCULOSIS (ATTESTED HERDS) SCHEMES

The number of Attested Herds, *i.e.*, herds officially certified as free from Tuberculosis as at 31st May, 1955, was as follows :—

ENGLAND	WALES	SCOTLAND	TOTAL (GREAT BRITAIN)
73,337	33,066	34,275	140,678